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NOTES

ON THE

DIAGNOSIS OF THE VARIOUS FORMS

OF

PARALYSIS OF THE MUSCLES OF EXTERNAL RELATION.

BY

JAMES ROSS, M.D., LL.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON; AND ASSISTANT PHYSICIAN TO THE MANCHESTER ROYAL INFIRMARY.

MANCHESTER: J. E. CORNISH.
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C. S. Sherrington

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PREFACE.

THIS little tract is intended to help the students of the Manchester School of Medicine in the comprehension of the diagnosis of the various forms of paralysis of the muscles of external relation, a subject on which I purpose to give a few clinical lectures during the forthcoming summer session. The simpler forms of paralysis are described in the briefest possible manner, and with the view of facilitating diagnosis they are so arranged that the points of agreement and difference between them can be readily apprehended. The morbid anatomy of such of those diseases as are caused by organic lesions of the nervous system is likewise described in very general terms, but it is believed with sufficient accuracy to give to the student an insight into the mechanism of their production. The time at my disposal will not permit me to enter on the discussion of the affections which have been named *mixed or indiscriminate* nervous diseases, and of which paralysis often forms a prominent symptom, but I have a profound conviction that those who possess an accurate knowledge of the simple or *system* diseases will not find much difficulty in unravelling the more complex affections. It will be my endeavour to supplement these notes in my lectures and to illustrate them by cases in such a way as to give to the student as accurate a knowledge as possible of this important subject.

JAMES ROSS.

MANCHESTER,
MAY 9TH, 1884.

AKINESIS OR PARALYSIS OF THE MUSCLES OF EXTERNAL RELATION.

By akinesis or paralysis of the muscles of external relation is understood the diminution or abolition of the power to contract the affected muscles by voluntary effort. The term *paresis* is used to denote the diminution of motor power. Some authors have endeavoured to restrict *paralysis* to its complete abolition, but this term will be employed here in a generic sense as embracing both conditions.

CLASSIFICATION.

Paralyses of the muscles of external relation are susceptible of being classified according to the nature, cause, and situation of the lesion, the distribution of the paralysis, and the functional disorders of the muscles which accompany paralysis of them. These divisions constitute the—I. pathological, II. etiological, III. topographical, IV. clinical, and V. physiological classification.

I. THE PATHOLOGICAL CLASSIFICATION.

The different varieties of paralysis are arranged, according to the *nature* of the lesion, into rheumatic, syphilitic, inflammatory, and other forms. They may also be divided into *organic* and *functional* lesions according as the morbid changes which underlie the paralysis are or are not capable of being recognised by our present means of research.

II. THE ETIOLOGICAL CLASSIFICATION.

The *functional* paralyses are not susceptible of being arranged according to the situation of the lesion, and they are therefore usually classified according to the cause of the affection. They are usually divided into (1) toxic, (2) febrile and post-febrile, (3) reflex, (4) post-epileptic, and (5) hysterical paralysis.

III. THE TOPOGRAPHICAL CLASSIFICATION.

The organic paralyses lend themselves readily to a classification according to the situation of the lesion. They may be divided into (I.) *myopathic* paralyses, or those in which the primary disease is situated in the muscles themselves, and (II.) *neuropathic* paralyses, or those in which the primary disease is localised in some part of the nervous system.

The *neuropathic* paralyses, with which we have to do chiefly here, may be sub-divided into (1) cerebral, (2) spinal, and (3) neural or peripheral paralyses, according as the lesion is situated in the brain, spinal cord, or peripheral nerves respectively. But although this division is very convenient, a much more important distinction is that which divides them into (1) *cerebro-spinal* and (2) *spino-peripheral* paralyses.

In the *cerebro-spinal* variety the lesion is situated either in the motor centres of the cortex of the brain or in the pyramidal tracts, the fibres of which connect these centres with the ganglion cells of the anterior grey horns of the spinal cord and their homologues in the medulla oblongata, pons, and crura cerebri.

In the *spino-peripheral* variety the lesion is situated in the anterior grey horns of the spinal cord and their upward continuations in the medulla oblongata, pons, and crura cerebri, or in the fibres of the peripheral nerves which connect the ganglion cells of these horns with the muscles. It will immediately be found that this topographical division corresponds more or less closely with the divisions of the physiological classification, and this constitutes its chief advantage.

It may here be noticed that when the lesion is restricted to one of the physiological tracts of the spinal cord the affection is called a *system-disease*, and when several of them are simultaneously implicated the affection is called a *mixed or indiscriminate* disease. Amongst the *mixed* diseases we shall also include complicated cases of cerebral paralysis.

IV. THE CLINICAL CLASSIFICATION.

Various names have been given to a paralysis according to its extent and distribution. The paralysis is sometimes limited to a single muscle or group of muscles, or all the muscles supplied by

a single nerve or plexus of nerves may be implicated; when all or almost all the muscles of a single extremity are paralysed, the condition is called a *monoplegia*. In other cases the paralysis affects both halves of the body symmetrically, and then it generally begins in the lower extremities, and spreads to the trunk and upper extremities. This is the usual form of paralysis which results from disease of the spinal cord, and is termed *paraplegia*. In other cases the paralysis affects the lateral half of the body, implicating the face, arm, and leg of the same side, and it is then termed *hemiplegia*. The lesion which causes this form of paralysis is usually situated in the opposite hemisphere of the brain; hemiplegia of spinal origin is named *hemiparaplegia*. When the upper and lower extremities on both sides are paralysed the condition, when due to spinal disease, has been named *paraplegia cervicalis*, and when caused by cerebral disease, *bilateral hemiplegia*, or *panplegia*. When the ocular or facial muscles on one side, and the limbs on the opposite side, are paralysed, the condition is called *crossed or alternate hemiplegia*.

V. THE PHYSIOLOGICAL CLASSIFICATION.

A much more important distinction than those depending upon the extent and distribution of the affection is that which divides the various forms into (1) *atrophic*, and (2) *spastic or spasmodic paralysis*. This division does not embrace every form of paralysis, inasmuch as in some paralytic affections the muscles neither undergo active wasting nor are affected by spasm. This distinction is, nevertheless, a very important one, and ought to be kept in view in the clinical examination of every case of paralysis. Both atrophic and spasmodic paralyses embrace some forms of myopathic paralysis, but when these are due to an organic disease of some part of the nervous system they correspond respectively to the spino-neural and cerebro-spinal paralyses of the topographical classification.

The different methods of classification are combined into one scheme in the following tables:—

TABLE I.

NEUROPATHIC PARALYSES.

A. ORGANIC PARALYSES.

I. SPINO-NEURAL OR ATROPHIC PARALYSES.

(I.) Neural or Peripheral Paralysis.

(II.) Spinal Atrophic Paralyses.
1. Acute Atrophic Spinal Paralysis of Infants.
2. Acute Atrophic Spinal Paralysis of Adults.
3. Paralysis Ascendens Acuta.
4. Chronic Atrophic Spinal Paralysis.
5. Peri-Ependymal Myelitis—Syringomyelia.
6. Progressive Muscular Atrophy.
7. Primary Labio-Glosso-Laryngeal Paralysis.
8. Ophthalmoplegia Externa.
9. Lead Paralysis (?).
10. (Pseudo-Hypertrophic Paralysis.)

II. CEREBRO-SPINAL OR SPASMODIC PARALYSES.

(I.) Spinal Spasmodic Paralyses. (PARAPLEGIÆ.)
1. Primary Lateral Sclerosis.
2. Compound Lateral Sclerosis.
   - *a.* Amyotrophic Lateral Sclerosis.
   - *b.* Combined Posterior and Lateral Sclerosis.
3. Secondary Lateral Sclerosis.
   - *a.* Compression Myelitis.
   - *b.* Transverse Myelitis.

(II.) Cerebral Paralyses. (HEMIPLEGIÆ.)
1. Tonic Spasm.
   - *a.* Early Rigidity.
   - *b.* Late Rigidity.
2. Clonic & Clonic Spasm.
   - *a.* Intermittent Tremor.
   - *b.* Choreiform Movements.
     - i. Præ-Hemiplegic Chorea.
     - ii. Post-Hemiplegic Chorea.
     - iii. Spastic Hemiplegia of Infancy.
3. Clonic Spasm
   - *a.* Continuous or Remittent Tremors.
   - *b.* Athetosis.
   - *c.* Post-Hemiplegic Hemiataxia.

III. MIXED PARALYSES.

B. FUNCTIONAL PARALYSES.

(1) Toxic Paralysis.
(2) Febrile and Post-Febrile Paralysis.
(3) Reflex Paralysis.
(4) Post-Epileptic Paralysis.
(5) Hysterical Paralysis.

TABLE II.

PARALYSES FROM ORGANIC DISEASE OF THE NERVOUS SYSTEM.

CLINICAL DIAGNOSIS.	TOPOGRAPHICAL DIAGNOSIS.
I. ATROPHIC PARALYSES.	**I. SPINO-NEURAL LESIONS.**
(I.) NEURAL or PERIPHERAL PARALYSES.	(I.) LESIONS OF PERIPHERAL NERVES.
(II.) SPINAL ATROPHIC PARALYSES.	(II.) LESIONS OF ANTERIOR GREY HORNS. (POLIOMYELOPATHIES.)
1. Acute Atrophic Spinal Paralysis of Infants.	Poliomyelitis Anterior Acuta Infantium.
2. Acute Atrophic Spinal Paralysis of Adults.	Poliomyelitis Anterior Acuta Adultorum.
3. Acute Ascending Paralysis.	Poliomyelitis Acuta.
4. Chronic Atrophic Spinal Paralysis.	Poliomyelitis Anterior Chronica.
5. Peri-Ependymal Myelitis. 6. Progressive Muscular Atrophy. 7. Primary Labio-Glosso-Laryngeal Paralysis. 8. Ophthalmoplegia Externa. 9. Lead Paralysis (?).	Degeneration of the Ganglion Cells of the Anterior Horns of the Spinal Cord and Motor Cells of the Medulla Oblongata.
10. (Pseudo-Hypertrophic Paralysis.)	(Primary Muscular Disease.)
II. SPASMODIC PARALYSES.	**II. CEREBRO-SPINAL LESIONS.** (PYRAMIDAL TRACT.)
(I.) SPINAL SPASMODIC PARALYSES.	(I.) LESIONS OF THE LATERAL COLUMNS.
1. Primary Spinal Spasmodic Paralysis.	Primary Lateral Sclerosis.
2. Compound Spinal Spasmodic Paralysis.	Amyotrophic Lateral Sclerosis. Combined Posterior and Lateral Sclerosis.
3. Secondary Spinal Spasmodic Paralysis.	Compression Myelitis. Transverse Myelitis.
(II.) CEREBRAL PARALYSES.	(II.) LESIONS OF THE CEREBRAL PYRAMIDAL TRACT AND MOTOR AREA OF CORTEX.
1. Ordinary Hemiplegia.	Lesions of Lenticular Nucleus. Area of Lenticulo-Striate Artery.
2. Alternate Hemiplegia.	Lesions of Crura and Pons.
3. Hemiplegia and Hemianæsthesia.	Lesions in Area of Opto-Striate Artery.
4. Hemiplegia, Hemianæsthesia and Hemianopsia. 5. Præ-Hemiplegic Chorea. 6. Post-Hemiplegic Chorea. 7. Athetosis. 8. Post-Hemiplegic Continuous Tremor and Hemiataxia.	Lesions in the Area of the Posterior External Optic Artery.
9. Spastic Hemiplegia of Infancy.	Unilateral Atrophy of the Motor Area of Cortex. Porencephalus.
10. Unilateral Convulsions and Hemiplegia.	Lesions of Motor Area of Cortex.

NEUROPATHIC PARALYSES.

ELECTRO-DIAGNOSIS.

FARADIC CURRENT.

Muscular contraction may be induced by the *direct* application of the faradic current to the muscles themselves, or *indirectly* through excitation of the motor nerves. Direct excitation of accessible muscles is best performed when the poles are applied over the points at which motor nerves enter the muscles, and these may be ascertained by reference to Ziemssen's diagrams. The faradic excitability of the muscles and nerves is normal or slightly increased in the various forms of the spasmodic paralyses, while it is diminished or completely abolished in the atrophic paralyses with the exception of the active stage of progressive muscular atrophy, in which it may be increased.

GALVANIC CURRENT.

The Law of Normal Contraction.

Let An = anode, Ca = cathode, C = contraction, c = feeble contraction, C′ = strong contraction, S = closure of current, O = opening of current, Te = tetanic contraction.

Weak currents produce .....Ca S C.
Medium ,, ,, ...Ca S C′, An S c, An O c.
Strong ,, ,, ...Ca S Te, An S C, An O C, Ca O c.

Deviations of this law may occur in disease by way of increase, diminution, or by changes in the quality of the various reactions.

1. *Simple Increase of the Excitability.*

Weak currents produce .....Ca S C′, An O C.
Medium ,, ,, ...Ca S Te, An O C′, Ca O C.
Strong ,, ,, ...Ca S Te, An O Te, Ca O C′.

2. *Simple Decrease of the Excitability.*

1st degree, strong currents produce...Ca S C, An S c, An O c.
2nd degree, ,, ,, ,, ...Ca S c.

3. *Qualitative Changes.*

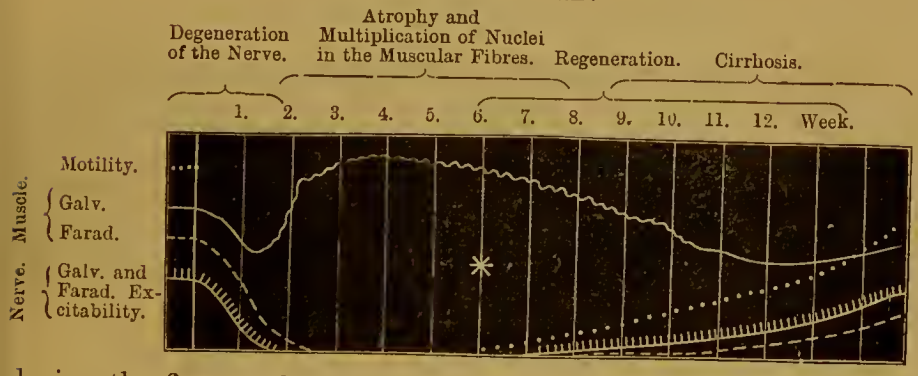
Weak currents produce during stage of increase ..... } Ca S C, An S Te, An O C, Ca O C′.
Medium currents produce during stage of gradual decrease ... } Ca S c, An S Te.
Strong currents produce during final stage prior to abolition ..... } An S c.

THE REACTION OF DEGENERATION.

The following diagrams, borrowed from Erb, represent graphically the general relations of motor power, electrical excitability, and structural changes of the nerves and muscles which are present in the different stages of paralysis. The first thick vertical line or ordinate indicates the sudden appearance of paralysis (▬), and the period of return of motor power is indicated by a (*), whilst the succeeding ordinates represent intervals of one or more weeks dating from the occurrence of the attack. The undulations in the line representing the galvanic excitability of the muscle indicate qualitative changes in the reactions. In the first degree of the reaction of degeneration (*Fig.* 1), the electrical excitability of both nerve and muscle falls

FIG. 1.

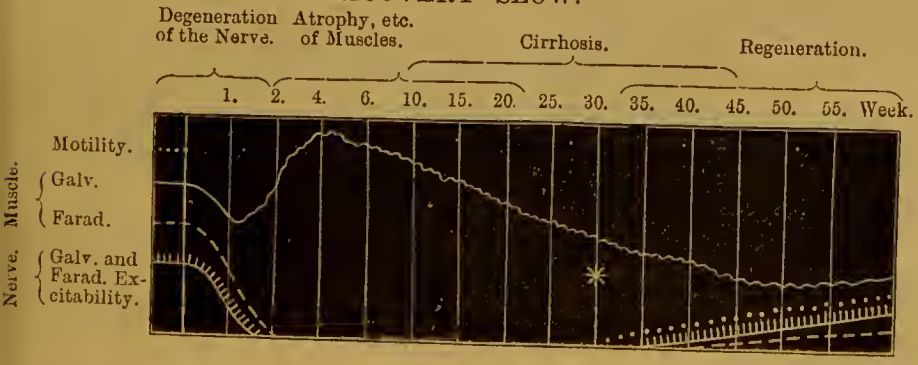
RECOVERY RAPID.



during the first week, the nerves lose all their electrical reactions during the second week, but the muscles lose only their faradic contractility during this period, whilst the galvanic excitability

FIG. 2.

RECOVERY SLOW.

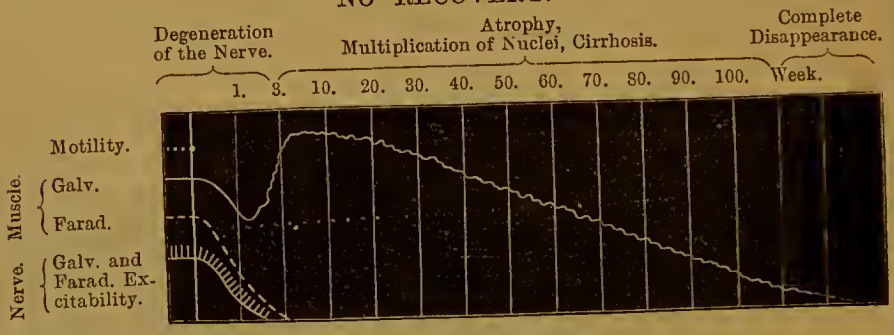


becomes greatly increased and manifests the qualitative changes already described. At the end of the sixth week there is a gradual return of motor power, and at the end of the seventh week there is a gradual return of the electrical reactions of the nerve and of the faradic contractility of the muscle, while the galvanic reactions of the muscle gradually sink, and the qualitative changes disappear until gradually the normal reaction is established.

In the second degree of the reaction of degeneration the faradic and galvanic excitability of the nerve does not appear until the thirtieth week (*Fig.* 2), while in the third degree (*Fig.* 3) the excitability of the nerve never returns, but the

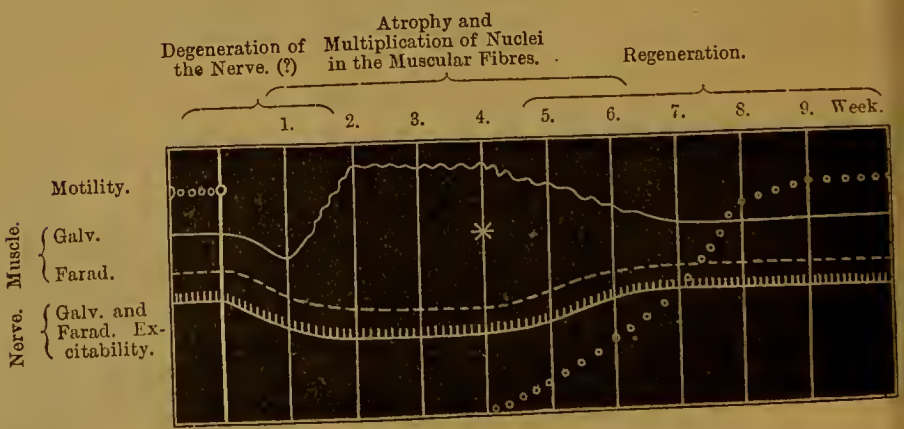
FIG. 3.

NO RECOVERY.



galvanic excitability of the muscle only becomes finally lost after a prolonged period, in some cases extending over a period of two years.

FIG. 4.



A "partial reaction of degeneration" (*Fig.* 4) has been described by Erb, in which the faradic and galvanic excitability of the affected nerve is diminished, but not abolished, the diminution being sometimes only to a slight degree. The faradic excitability of the paralysed muscle undergoes a diminution corresponding to that of the nerve, but the galvanic excitability of the muscles manifests the quantitative and qualitative changes which are so characteristic of the severer form of the reaction of degeneration.

A. ORGANIC PARALYSES.

I. ATROPHIC PARALYSES.

(1.) Neural or Peripheral Paralysis.

(1) *Traumatic Division and Compression of Nerves.*—The paralysed muscles are grouped according to the anatomical distribution of the nerve, and not according as they are associated in their functions, and the motor phenomena are accompanied by anæsthesia in the area of distribution of the sensory branches of the nerve. The affected muscles undergo atrophy, and manifest the reaction of degeneration. The cicatrix of a wound or evidences of a tumour can generally be discovered in the track of the nerve, and trophic affections of the skin, nails, and joints are present.

(2) *Acute and Chronic Neuritis.*—Symptoms of motor irritation, such as tremors, sudden contractions, or persistent contractures, are present in the early stage of the disease, and these are succeeded by paralysis. Reflex spasms may also be present. Continuous pain, with paroxysmal exacerbations, is felt in the area of the sensory branches, and this may be accompanied by various paræsthesiæ and varying degrees of anæsthesia. The affected nerve may be felt as a thick cord, or as fusiform swellings *(neuritis nodosa)*, and it is very sensitive to pressure, which also gives rise to eccentric pains and formication. The other symptoms are the same as in traumatic division of the nerves.

(3) *Injuries of Nerve Roots.*—The roots of some of the cranial nerves may be ruptured in fractures of the skull and compressed

by the growth of tumours. The spinal roots may also be compressed by the growth of tumours, and by inflammatory thickenings of the membranes, as in pachymeningitis.

Rupture of the Roots of the Brachial Plexus is caused by severe strain upon the shoulder-joint. It gives rise to atrophic paralysis of the muscles supplied by the brachial plexus, a widely distributed anæsthesia of the skin of the upper extremity, and oculo-pupillary phenomena on the side of the injury caused by implication of the fibres issuing from the cilio-spinal region of the cord.

(4) *Progressive Multiple Neuritis.*—The affection is ushered in by fever and tingling pains in the hands and feet. Pressure over the affected nerves, which are often swollen, causes great pain, that radiates towards the periphery. The skin and muscles in the territory of the affected nerves are likewise very sensitive to pressure, and the slightest touch or passive movements give occasion to loud complaints from the patient. This hyperæsthesia is accompanied by well-marked anæsthesia of certain forms of sensibility. Motor paralysis soon manifests itself; it begins in the small muscles of the hands and feet, and successively invades the larger muscles of the limbs and those of the trunk, while in some cases those of the bladder and rectum are involved, and death may result from implication of the intercostal and bulbar nerves. The affected muscles rapidly waste and give the reaction of degeneration. The hands assume distorted positions as in progressive muscular atrophy, and trophic affections of the skin, nails, and hair have been observed. The affection, which begins as an acute disease, soon becomes chronic, and complete or partial recovery may take place in from nine to eighteen months from the commencement.

Morbid Anatomy.—The chief lesions have been found in the peripheral nerves, while the spinal cord and brain remain healthy. The morbid changes consist of destruction of the nerve fibres, along with thickening of the perineurium and an accumulation of fat cells between the bundles.

(5) *Diphtheritic Paralysis.*—The affection usually begins by paralysis of the veil of the palate, to which may soon be added paralysis of the abductors of the vocal cords, and of the tongue, lips, and masticatory muscles. The ocular muscles are the next to be attacked, and the patient is then unable to read small print from paralysis of accommodation; or there is mydriasis, from paralysis of the sphincter of the iris; or diplopia, strabismus, or ptosis, from paralysis of one or more of the external muscles of the eye. The patient may have a vacant and idiotic expression from feebleness of the facial muscles, but complete paralysis of them is rare. The paralysis now extends to the muscles of the limbs, and the inferior extremities are usually the first to be attacked; in most cases the muscles of the superior extremities are subsequently implicated, and ultimately the muscles of the trunk and neck, including the muscles of respiration, may be affected. Sensory disorders always accompany the loss of motor power in this form of paralysis. The mucous membrane of the soft palate and pharynx is often anæsthetic; the patient complains of numbness and tingling pains in the hands and feet; the tactile sensibility is diminished in the extremities; and they are sometimes completely anæsthetic. Even the functions of the special senses are sometimes diminished. The reflex action of the palate is diminished or lost at an early period of the disease, and the tendon-reactions are likewise lost, but the condition of the cutaneous reflex actions in the extremities have not been accurately observed. The electrical excitability of the affected nerves and muscles is said by Duchenne to be retained, but recent observations seem to show that the muscles manifest the partial reaction of degeneration (*Fig.* 4). In some cases the heart becomes implicated in the paralysis; the pulse is slow, irregular, and feeble, and there are paroxysms of palpitation accompanied by præcordial anxiety and dyspnœa, while death by syncope is by no means uncommon. Obstinate constipation from paralysis of the muscular coats of the bowel is a frequent symptom, and the bladder and rectum are occasionally involved. It is calculated that twelve per cent of all cases terminate fatally; of the remainder by far the greater proportion recover completely, while in a few only is the recovery imperfect.

Morbid Anatomy.—There are good grounds for believing that the morbid changes in this affection consist of a parenchymatous inflammation or degeneration of the peripheral nerves similar to that which occurs in progressive multiple neuritis.

(II.) Spinal Atrophic Paralyses.
Poliomyelopathies.

Diseases of the grey matter of the anterior horns of the spinal cord are characterised by an atrophic paralysis of the muscles supplied from the seat of the lesion, and by the absence of any pronounced sensory disturbances or disorder of the functions of the bladder and rectum.

(1) *Acute Atrophic Spinal Paralysis of Infants.*—The affection begins, in the majority of cases, between the ages of six months and three years with a febrile attack, and, occasionally, convulsions, but in some cases all general symptoms are absent. The paralysis becomes suddenly developed and reaches its maximum of extent and intensity in a few hours, or at most in a few days from the commencement; one or two or all of the limbs are found to hang down in a relaxed and powerless condition, and the muscles of the neck, face, or tongue have occasionally been implicated. The sensibility is almost entirely unaffected throughout the whole progress of the disease, and the sphincters are intact. The affected muscles undergo rapid atrophy, and manifest the first, second, or third kind of the "reaction of degeneration" (*Figs.* 1, 2, and 3) according to the degree of paralysis and the prospect of recovery. The bones and tendons undergo trophic changes, but the skin and its appendages escape, but the temperature of the affected limbs sinks considerably below normal. After the first few days gradual recovery takes place in some of the paralysed muscles; the improvement proceeds most actively during the first four to eight weeks, but it may continue in fair activity for nine months, and may go on slowly for one or two years. In some few cases complete recovery may take place (temporary spinal paralysis), but as a rule one or several groups of muscles remain permanently paralysed, giving rise, according to the extent and degree of loss of motor power,

to such deformities as the various forms of talipes, pes cavus, genu recurvatum, claw hand, flattening of the shoulder and lordosis. The bones of the affected extremities are often arrested in their development, the joints become unusually moveable and deformed owing to the disappearance of the articular extremities and relaxation of ligaments, but the nutrition of the skin is unaffected.

(2) *Acute Atrophic Spinal Paralysis of Adults.*—This form of paralysis corresponds to the acute atrophic spinal paralysis of infants, and differs from it only in the facts that it begins in adult age instead of in infancy, that the onset is not marked by initial fever or convulsions, and that the resulting deformities are less pronounced.

Morbid Anatomy.—In the atrophic paralysis of infants and adults the lesion is more or less limited to the anterior grey horn of the spinal cord. Some pathologists believe that the

FIG. 5.

FIG. 5 (from Charcot). *Transverse Section of the Spinal Cord* taken from the cervical region of a woman, aged fifty years, who died in the Salpétrière, of general paralysis of the insane, and who was the subject of infantile spinal paralysis of the right superior extremity. There was fibroid atrophy of the right anterior cornu, and atrophy of all the white columns of the corresponding side.

morbid changes begin by a hæmorrhage, while others are of opinion that the disease is of the nature of an acute inflammation. In chronic cases the anterior horn has been found in a state of sclerosis (*Figs.* 5 and 6), with more or less complete destruction of the multipolar ganglion cells. The anterior roots originating from the seat of the lesion in the cord have also been found thin and atrophied. The muscles undergo profound changes, which need not be described here.

FIG. 6.

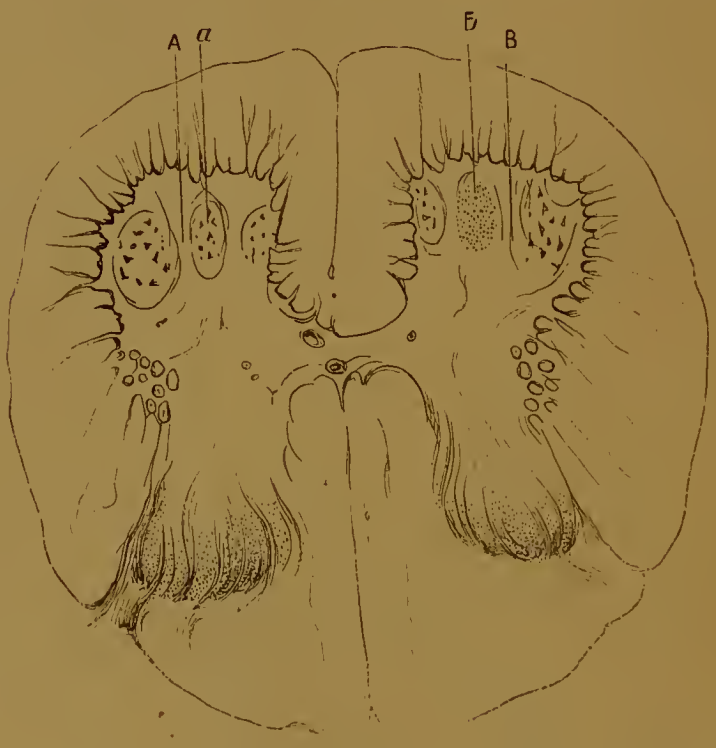


FIG. 6 (from Charcot). *Section of the Spinal Cord in the Lumbar Region, from a case of infantile paralysis.*—A, Left anterior cornu, healthy; *a*, Healthy median group of ganglion cells. B, Right anterior cornu; *b*, Median group of ganglion cells. The cells are destroyed, and the group is represented by a patch of sclerosis.

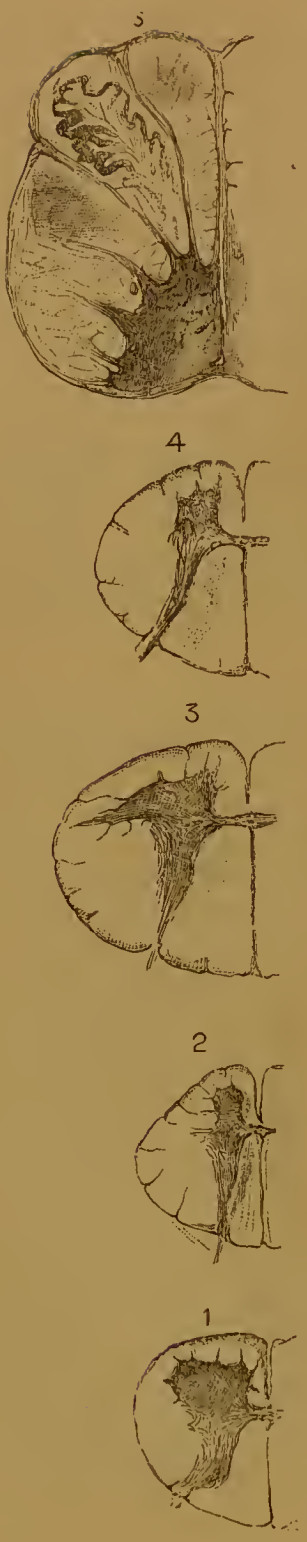
(3) *Paralysis Ascendens Acuta—Landry's Paralysis.*—It is characterised by a motor paralysis which begins in the lower extremities and spreads pretty rapidly over the trunk to the upper extremities, ascending until the medulla oblongata is involved. The general sensibility and the functions of the

bladder and rectum are either unaffected or only slightly implicated, and there is no decided atrophy of the paralysed muscles and no alteration of their electrical excitability, but the patellar-tendon reactions are lost at an early period. The disease may terminate fatally in two or three days, but it lasts occasionally from two to four weeks, the average of fatal cases being from eight to twelve days. The disease may end in recovery; its progress may become arrested in any part of its course, the parts last attacked are the first to show signs of improvement, and after a long period of debility recovery becomes complete.

Morbid Anatomy.—In most of the cases hitherto examined no morbid changes have been discovered in the spinal cord, peripheral nerves, or other parts of the nervous system, on microscopical examination. Déjerine and Goetz found changes in the anterior nerve roots in one case, and evidences of acute myelitis were observed by Leyden in another.

(4) *Chronic Atrophic Spinal Paralysis.*—The paralysis usually begins in the lower extremities and gradually progresses upwards until the muscles of the trunk and upper extremities are involved, although occasionally the upper extremities begin first, and the paralysis then pursues a descending course. The affected muscles manifest at an early period the reaction of degeneration; they undergo rapid atrophy, and the hands and feet assume characteristically deformed positions. The cutaneous reflexes and the tendon reactions are lost at an early period, but the general sensibility and the functions of the bladder and rectum are unaffected. The small joints of the extremities, and especially those of the hands, sometimes become swollen, as in rheumatic arthritis, but the nutrition of the skin is unaffected. The paralysis may implicate the bulbar nuclei in its ascending course, and death may take place from asphyxia or from syncope, but the disease often becomes arrested, and gradual improvement takes places, the motor power returning in the reverse order to that in which it was lost. Recovery is so slow that it is only after the lapse of months that the patients are able to feed themselves, and it often remains incomplete in the lower extremities.

Fig. 7.



Morbid Anatomy.—The spinal cord was examined by Cornil and Webber, and the chief changes were found in the anterior grey horns of the spinal cord, and consisted of disappearance and atrophy of the ganglion cells, disappearance of their processes, thickening of the walls of the blood-vessels, and exudation of white blood corpuscles into the perivascular spaces. In a case reported by myself the ganglion cells had almost completely disappeared (*Fig.* 7).

(5) *Peri-Ependymal Myelitis—Syringomyelia.*—The symptoms are variable, but they generally consist of a motor paralysis which may begin in the upper or lower extremities and pursue a descending or an ascending course. The paralysed muscles become atrophied and manifest the reaction of degeneration. The patient may complain of vague pains along the vertebral column, and occasionally there may be an extensively diffused anæsthesia of the lower extremities, but the sphincters remain unaffected. The course of the affection is slow, and it may be temporarily arrested, or even complete recovery may take place.

Fig. 7 (Young). *Transverse Sections of the Spinal and Medulla Oblongata at different levels, from a case of chronic atrophic spinal paralysis, showing the disappearance of the ganglion cells.*—1, Middle of the lumbar enlargement; 2, Middle of the dorsal region; 3, Middle of the cervical enlargement; 4, Section on a level with the origin of the second cervical nerve; 5, Section of the medulla oblongata on a level with the middle third of the olivary body.

Morbid Anatomy.—The morbid process begins in the neuroglia surrounding the central canal, and the central column becomes converted into a solid mass of fibroid tissue which grows from within outwards and invades the remaining parts of

FIG. 8.

FIG. 8 (from Leyden). *Transverse Section of the Spinal Cord from the Middle of the Cervical Enlargement, from a case of Syringomyelia, showing a cavity behind the posterior commissure, and destruction of a large portion of the ganglion cells of the anterior grey horns.*

the grey matter. The central core may be solid throughout (*peri-ependymal myelitis*) (*Fig.* 8), but in most cases a portion of the interior undergoes softening, and a cavity is formed which is filled with serous fluid (*syringomyelia*) (*Fig.* 9). This cavity

FIG. 9.

FIG. 9 (after Gull). *Transverse Section of the Cervical Enlargement of the Spinal Cord, showing a central cavity, which has destroyed considerable portions of the anterior grey horns.*

sometimes extends the whole length of the cord, while at other times it is only a few lines in length; in transverse area it is sometimes large enough to admit the tip of the finger, while at other times it is only large enough to be visible to the naked

eye. It is most usually situated, not in the central canal, but in that part of the posterior column which adjoins the posterior column.

(6) *Progressive Muscular Atrophy.*—This disease is a progressive atrophy with paralysis of the voluntary muscles, which pursues a chronic course, and successively attacks individual muscles and groups of muscles, the affected muscles being the

FIG. 10.

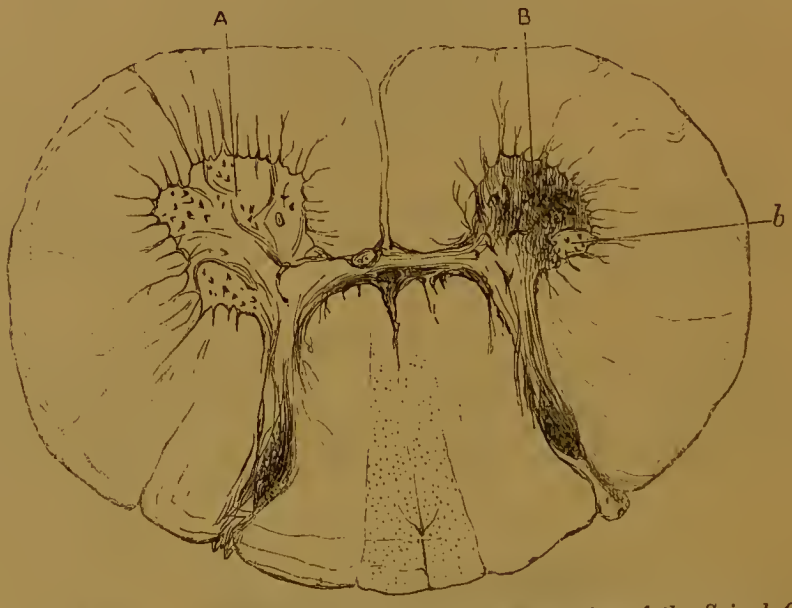


FIG. 10 (Charcot). *Transverse Section of the Cervical Region of the Spinal Cord, from a case of progressive muscular atrophy.*—A, Left anterior grey horn; the ganglion cells have persisted, but are much altered in appearance. B, Right anterior grey horn, almost complete atrophy of the cells, one group only (*b*) having persisted.

subjects of fibrillary contractions during the active stage of the disease. The muscular wasting precedes the paralysis, and so long as any muscular fibres are left they can be made to contract by voluntary effort. The electrical reactions of both nerves and muscles remain normal for a long time, and it is only after the muscles have undergone a high degree of atrophy that the "reaction of degeneration" is obtained. The reflex actions are occasionally exaggerated in the early stage of the disease, but they become diminished as the disease advances. The sensibility is not much affected; the muscular atrophy is sometimes preceded

by paroxysms of pain, and in the later stages of the disease a moderate degree of anæsthesia may be present in the hands and feet. The temperature of the affected extremities may be increased in the earlier stages of the disease, but in the later stages it is diminished. Arthritis nodosa and oculo-papillary symptoms are occasionally present. The disease usually begins in the upper extremities, and the muscles of the thenar and hypothenar eminences, and the interossei, or the muscles of the

Fig. 11.

Fig. 11 (from Leyden). *Portion of the Grey Substance on the floor of the fourth ventricle on a level with the middle of the Hypoglossal Nucleus, from a case of Progressive Muscular Atrophy with Bulbar Paralysis, showing the destruction of the Ganglion Cells of the nuclei of the Hypoglossal and Pneumogastric Nerves (NH). R*, Median raphé, *H*, *H*, Fibres of the hypoglossal nerves. The accessory neuclei have evidently disappeared.

shoulder are, as a rule, the first to be attacked. The unantagonised action of the healthy muscles, combined with the wasting of the diseased ones, gives rise to characteristic deformities. Recovery occurs occasionally, but the disease often terminates fatally after an average duration of five years.

Morbid Anatomy.—The essential morbid lesion consists of a chronic and progressive atrophy of the ganglion cells of the anterior grey horns of the spinal cord (*Fig.* 10). The anterior roots of the nerves are likewise atrophied, and the muscles

undergo progressive changes, which ultimately leads to atrophy of the muscular fibres.

(7) *Primary Labio-glosso-Laryngeal Paralysis.*—It consists of a progressive paralysis and atrophy of the muscles of the tongue, lips, soft palate, pharynx, and larynx, giving rise to difficulties of articulation, deglutition, and phonation. The general sensibility is not affected, but there may be some degree of anæsthesia of the soft palate, fauces, and larynx, and the reflex contractility of the palate is often found diminished at an early period of the disease. This affection appears to be essentially the same disease as progressive muscular atrophy; the chief difference being that in the former the affected muscles are innervated by the bulbar nerves, while in the latter they are innervated by spinal nerves. The bulbar affection is often complicated by a progressive muscular atrophy of the muscles of the upper extremities.

Morbid Anatomy.—The essential lesion consists of a progressive atrophy of the ganglion cells of the bulbar nuclei (*Fig.* 11) with atrophy of the motor roots of the cranial nerves.

(8) *Ophthalmoplegia Externa.*—This affection consists of a progressive paralysis of the ocular muscles, which appears to run a course more or less similar to the progressive paralysis of the lips, tongue, and soft palate, just described. It generally begins by a drooping of the eyelids, and soon afterwards all the muscles of the eyeballs manifest signs of weakness, although all of them are not affected simultaneously or to the same degree. The paralysis gradually increases until ultimately both globes are completely immoveable. The fifth and seventh nerves are often implicated, and blindness with white atrophy of the optic nerves has been observed in about one-third of the recorded cases. The symptoms are occasionally associated with atrophy of the muscles of the back and shoulders, and more frequently with some of the characteristic signs of locomotor ataxia, such as lancinating pains and other sensory disorders in the lower extremities, absence of the patellar-tendon reaction, reflex immobility of the pupil swaying movements on closing the eyes, or ataxic gait.

Morbid Anatomy.—Dr. Gowers found in one case degeneration of the roots of the ocular motor nerves, and disappearance of the cells from their nuclei of origin. The affection would appear to be allied to bulbar paralysis. Syphilis is the most frequent cause of the affection.

(9) *Lead Paralysis.*—Almost all the muscles of the body may be affected in lead poisoning, but the extensor muscles of the forearm are attacked by preference, while the supinator longus is spared until a comparatively late period of the disease. The paralysed muscles undergo rapid atrophy, and lose their faradic contractility before voluntary power is completely abolished, while the reaction of degeneration appears in them at an early period. The occupation of the patient and the blue line on the gums afford valuable diagnostic signs. Cutaneous hyperæsthesia followed by anæsthesia is an occasional symptom of chronic lead poisoning, and arthralgia is frequently present. The functions of the special senses are occasionally diminished.

Morbid Anatomy.—Morbid changes have been found in the intermuscular nerve fibres, the nerves themselves, and in the grey matter of the anterior horns of the spinal cord, and it is therefore somewhat doubtful what position is to be assigned to this form of paralysis in our classification. The fact that the muscles are paralysed according as they are associated in their functions, and not according to the anatomical distribution of any peripheral nerve, induces us to place lead paralysis provisionally amongst the spinal atrophic paralyses.

(10) *Pseudo-hypertrophic Paralysis.*—This affection begins, as a rule, in early childhood as a progressive paralysis, associated with atrophy of some of the muscles and apparent increase in the volume of others. The paralysis begins in the muscles of the lower extremities, and extends to the erector muscles of the spine and the muscles of the upper extremities, those about the shoulder and upper arm being implicated, whilst, with the exception of the supinator longus, the muscles of the forearm and hand are always spared. The most characteristic features of this affection are large calves of the legs and slender thighs, a peculiar

deformity of the foot which makes it necessary for the patient to balance himself on his toes, a dorsal eurvature named *lordosis*, a straddling gait, and a peculiar mode of aseending a stair and attaining the ereet posture. The disease is generally fatal at puberty, although some patients may live to advanced age.

Fig. 12.

Fig. 12 (Young). *Muscular Fibres in various stages of degeneration, from a case of Pseudo-hypertrophic Paralysis.*—*a*, Muscular fibre only slightly changed, showing increase of the muscle corpuscles, and indistinctness of the transverse striation in certain parts of its length; *b*, the same as *a*, but more atrophied; *c*, muscular fibre greatly atrophied, and presenting nuclei at intervals; *d*, atrophied muscular fibre, with its transverse striation unusually distinct; *e*, atrophied fibre surrounded by a fibrillated connective tissue rich in nuclei; *f* and *g*, muscular fibres from the erector spinæ, which manifested the greatest changes to the naked eye. These fibres appear to have undergone a hyaline change, but their transverse striation is still faintly visible. The fibres often tapered to a point, sometimes at one, and sometimes at both ends.

Morbid Anatomy.—This disease is a purely museular affeetion, and in several marked eases the spinal eord and nerves have been found healthy. The morbid ehange begins in the eonneetive tissue whieh separates the museular bundles from one another, and whieh passes between the museular fibres themselves. This ehange eonsists of a large deposit of fat eells in the eonneetive tissue, whieh eauses the musele as a whole to be greatly

increased in volume, although the muscular fibres become atrophied at an early period of the disease. The different changes which the muscular fibres themselves undergo are shown in *Fig.* 12.

II. SPASMODIC PARALYSES.

(I.) Spinal Spasmodic Paralyses.

1. *Primary Lateral Sclerosis.*

Begins with paresis of the lower extremities, which slowly increases in intensity, and may after a long time invade the upper extremities also. The affected muscles give normal or only slightly diminished electrical reactions, they are tense and offer resistance to passive movements of the various joints, and in the later stages of the disease the limbs become the subjects of contractures. The patient has a peculiar "waddling" and stiff gait. The cutaneous reflexes and tendon-reactions are exaggerated, but there is an entire absence of sensory and nutritive disorders, and the functions of the bladder and rectum are unaffected.

Fig. 13.

Fig. 13 (after Charcot). *Attitude of the Hand and Forearm in Amyotrophic Lateral Sclerosis.*

2. *Compound Lateral Sclerosis.*

(*a*) *Amyotrophic Lateral Sclerosis.*—The disease begins by a motor weakness of the upper extremities, accompanied by a uniformly diffused muscular wasting. Muscular tension and contractures are soon superadded, and the hand after a time assumes a characteristic deformity (*Fig.* 13).

After a period of from two to six months the lower extremities become affected by paralysis with muscular tension, contractures, and increase of the cutaneous reflexes and tendon-reactions. The third stage of the disease is characterised by the appearance of a progressively increasing paralysis of the muscles of the tongue, lips, pharynx and larynx, and by disturbances of circulation and respiration, which lead to death on an average from one to three years from the commencement.

(*b*) *Combined Sclerosis of the Posterior and Lateral Columns.* In this affection the paralysis, muscular tension, and contractures which characterise primary lateral sclerosis are present in the lower extremities, but the patellar-tendon reactions instead of being exaggerated are absent.

3. *Secondary Lateral Sclerosis.*

Lateral sclerosis occurs as a secondary affection in *transverse and compression myelitis* and in various diseases of the medulla oblongata, pons, and brain. The symptoms are the same, so far as the paralysis is concerned, as those of the primary variety, but in addition to the motor phenomena caused by sclerosis of the

FIG. 14. FIG. 15. FIG. 16.

FIGS. 14, 15, and 16 (after Charcot). *Transverse Sections of the Spinal Cord, from the middle of the cervical enlargement, middle of the dorsal region, and middle of the lumbar region respectively*, showing primary lateral sclerosis of the cord, or secondary to a lesion high up in the cord or medulla oblongata.—A, A, A, Degenerated pyramidal tracts.

lateral columns there are various sensory phenomena, and disorder of the functions of the bladder, rectum, and other organs, caused by the primary lesion.

Morbid Anatomy.—The lesion in primary lateral sclerosis consists of a symmetrical sclerosis of the lateral columns (*Figs.* 14, 15, and 16), and also of the columns of Türck (not represented in the Figures). In the amyotrophic variety

symmetrical sclerosis of the antero-lateral columns of the cord and of the anterior pyramids of the medulla oblongata is accompanied by degenerative atrophy of the anterior grey horns and loss of the ganglion cells of the cord and of the motor nuclei of the medulla oblongata. In combined sclerosis the posterior columns are implicated in the disease along with the lateral columns, while in secondary spinal sclerosis the disease of the lateral columns is caused by a transverse myelitis, the compression of a tumour, or some other lesion.

(II.) Cerebral Paralyses.

1. *Ordinary Hemiplegia.*

Hemiplegia consists of paralysis of one-half of the body, but many of the muscles are either not implicated or are only temporarily weakened. The paralysis is, as a rule, limited to the arm, leg, and part of the face.

2. *Alternate Hemiplegia.*

In this form the face or the ocular muscles supplied by the third nerve are paralysed on one side of the body, and the limbs on the opposite side.

3. *Hemiplegia and Hemianæsthesia.*

In this variety the paralysis of one-half of the body is accompanied by a hemianæsthesia of the same side, including every form of cutaneous and muscular sensibility, as well as the special senses. When hemiplegia is accompanied by hemianæsthesia, the leg is generally more profoundly affected than the arm.

4. *Hemiplegia, Hemianæsthesia, and Hemianopsia.*

In this form the motor paralysis and loss of sensation are accompanied by a homonymous lateral hemianopsia. In such cases the motor paralysis is usually slight in degree.

5. *Hemiplegic and Post-Hemiplegic Spasms.*

In hemiplegia the paralysis is accompanied by spasm of the affected muscles. These may be either, (*a*) tonic, (*b*) combined tonic and clonic, and (*c*) clonic spasms. In addition we must specially consider (*d*) spasm as occurring in the hemiplegia of infancy.

(*a*) *Tonic spasm* may be divided into (i.) *early* and (ii.) *late rigidity.*

(i.) *Early rigidity* consists of a tonic spasm of the paralysed muscles, which may occur either at the time of the hæmorrhage, or a few days subsequently during the inflammatory reaction.

(ii.) *Late rigidity* corresponds in its essential features to the spasmodic rigidity of primary lateral sclerosis, and is accompanied by exaggeration of the tendon-reactions.

(*b*) *Combined Clonic and Tonic Spasm.*—At first the spasm is fixed and tonic, like that of late rigidity; but after a time some of the muscles implicated become the subjects of clonic spasm. These spasms may be divided into two varieties, (i.) *intermittent tremor* and (ii.) *choreiform movements.*

(i.) *Intermittent Tremor.*—The tremor is induced when the muscles are put upon the stretch by passive movements, or by any attempt at voluntary action.

(ii.) *Choreiform Movements.*—These movements may occur either before or after an apoplectic attack, and are therefore divisible into *pre-hemiplegic* or *post-hemiplegic chorea.* The movements induced by these spasms are of wider range than those of hemiplegic tremor, being in this respect like the movements of chorea.

(*c*) *Clonic Spasms.*—These are unaccompanied by tonic contractions, and are divisible into (i.) *continuous or remittent tremor*, (ii.) choreiform movements (*athetosis*), and (iii.) jerking movements on voluntary effort (*hemiataxia*).

(i.) *Continuous or Remittent Tremor.*—The tremor in this variety remits during sleep, but is continuous during waking hours, as in paralysis agitans.

(ii.) *Athetosis.*—In athetosis the fingers and toes, and occasionally the hands, feet, neck, and face, are maintained in continuous slow movement, and are made to assume various distorted positions. The motor disorder is generally associated with some degree of hemianæsthesia.

(iii.) *Hemiataxia.*—In this variety there is an absence of permanent rigidity or spontaneous spasm, but the affected extremity is subject to disorderly or incoordinate movements on voluntary effort with closed eyes.

(*d*) *Spastic Hemiplegia of Infancy.*—These may be divided into (i.) *acquired,* and (ii.) *congenital hemiplegia.* Closely allied to them is (iii.) *double athetosis,* which is generally a congenital affection and associated with idiocy.

(i.) *Acquired Spastic Hemiplegia of Infancy.*—The disease begins at birth, or between that date and four or five years of age. The child is unconscious and convulsed for two or three days after birth (asphyxia neonatorum), or, if attacked after birth, it is seized with unconsciousness and convulsions which last from a few hours to a few days. The convulsions are limited to, or at least more pronounced on, one side of the body, and subsequently this side is found to be paralysed. In the cases which recover, the hemiplegia pursues the usual course, contractures become established, and choreiform movements may or may not make their appearance, but when they once supervene they remain permanent. The paralysed half of the body becomes arrested in its development as compared with the opposite side, the bones being smaller in all their dimensions. The intellectual faculties suffer to some extent, and the patient is more or less idiotic. At from seven to fifteen years of age epileptic attacks supervene; these are at first limited to the paralysed side of the body, and are not attended by decided loss of consciousness. In old-established cases the convulsions may become general, but they manifest a unilateral character at the commencement of the attack, and the patient usually describes a unilateral aura.

(ii.) *Congenital Spastic Hemiplegia of Infancy.*—The forms of paralysis which come under this category are very numerous and variable, and they are generally accompanied by imbecility or idiocy. Sometimes the

FIG. 17.

FIG. 18.

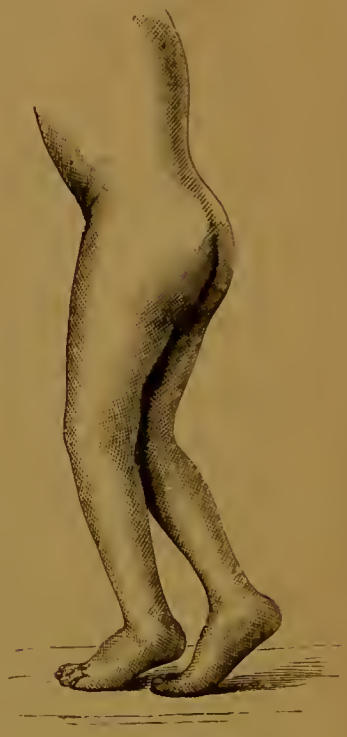


FIG. 19.

FIG. 19. *Portrait of a case of double athetosis with aphonia, showing the contortions of the hands and face.*

paralysis is limited to one-half of the body, while at other times both sides are affected. There are good grounds for believing that many cases of congenital paralysis (*Figs.* 17 and 18) limited to the lower extremities are not to be regarded as true spinal paraplegiæ but as examples of double hemiplegiæ.

FIG. 20.

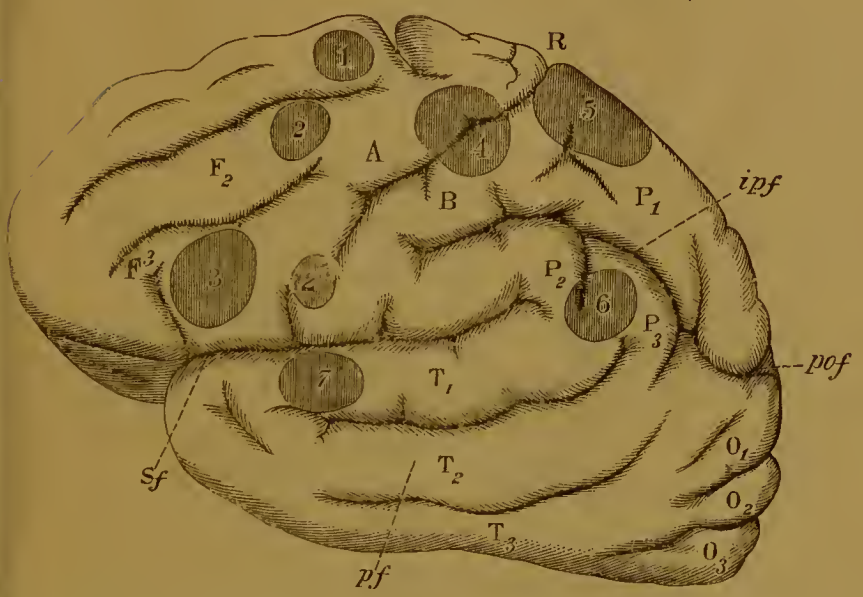


FIG. 20. *External Convex Surface of the Human Brain.—Fissures:* R, Fissure of Rolando; *Sf*, Fissure of Sylvius; *pf*, Parallel fissure; *ipf*, Interparietal fissure; *pof*, External parieto-occipital fissure. *Convolutions and Lobules:* A, Ascending frontal; B, Ascending parietal convolutions; F_1, F_2, F_3, First, second, and third frontal convolutions; P_1, Superior parietal lobule; P_2, Supra-marginal gyrus; P_3, Angular gyrus; O_1, O_2, O_3, First, second, and third occipital convolutions; T_1, T_2, T_3, First, second, and third temporo-sphenoidal convolutions. *Motor Centres:* 1, Movements for rotation of head and neck; 2, Movements of the upper facial muscles; 2′, Movements of the lower facial muscles; 3, Movements of the tongue and jaws; 4, Movements of superior extremity; 5, Movements of inferior extremity; 6, Movements of the ocular muscles; Movements in relation with the sense of hearing.

(iii.) *Bilateral Athetosis.*—This affection is generally of congenital origin, and is always associated with more or less idiocy, articulatory disorders and disturbances of speech (aphasia) being common. Both hands, and frequently the toes, are subject to choreiform spasms, and the face is likewise more liable to be implicated in the spasm than in the unilateral disease (*Fig.* 19). Sensory disorders are wanting.

6. *Unilateral Convulsions and Hemiplegia.*

Irritative lesions of the cortex give rise to unilateral convulsions or monospasms. The clinical varieties of unilateral convulsions may be divided into (*a*) crural, (*b*) brachial, (*c*) facial, (*d*) oculo-motor, and (*e*) masticatory monospasm or protospasm, according

FIG. 21.

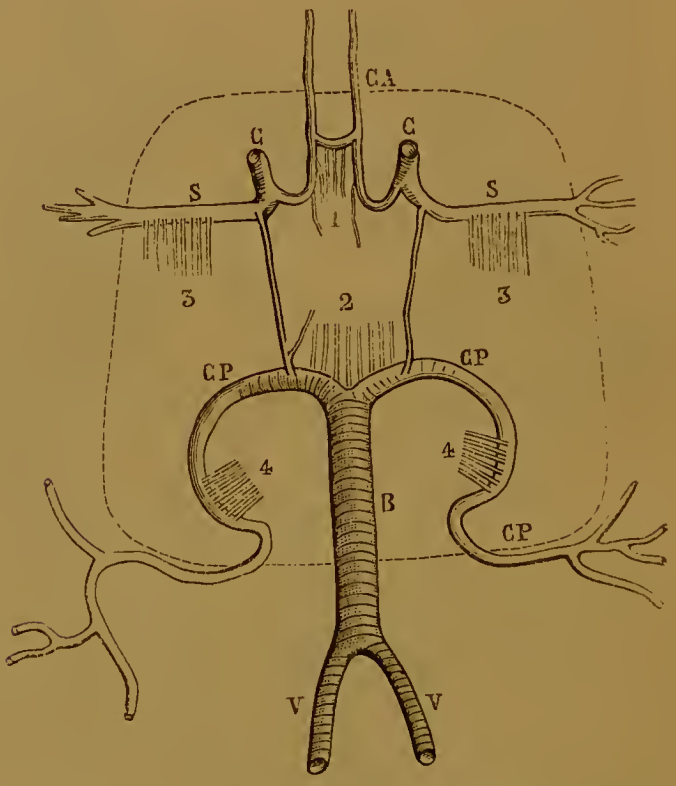


FIG. 21 (after Charcot). *Diagram of the Distribution of the Vessels at the base of the Cerebrum.*—CA, Anterior cerebral artery; S, S, Sylvian arteries; V, V, Vertebral arteries; B, Basilar; CP, CP, Posterior cerebral arteries; 1, 2, 3, 3, 4, 4, Groups of nutritive arteries. The line ---- limits the ganglionic vascular area.

as the spasm is limited to or begins in the muscles of the leg, arm, face, those which produce conjugated deviation of the eyes with rotation of the head and neck, and those of the jaw respectively. The muscles which were the subjects of spasm during the attack are more or less paralysed subsequently.

Morbid Anatomy.—Hemiplegia is caused by injury of the motor centres of the cortex (*Fig.* 20), or of the fibres which connect them with the spinal cord and which pass through the centrum semiovale, the internal capsule, crusta, pons, anterior pyramids of the medulla oblongata, to reach the antero-lateral columns of the spinal cord. As hemiplegia is usually caused by rupture or occlusion of one of the cerebral vessels, it is necessary to know accurately their distribution (*Figs.* 21 and 22). Of all the arteries of the brain, the

FIG. 22.

FIG. 22 (from Duret). *Transverse Section of the Cerebral Hemispheres, about* 1 *cm. behind the Optic Commissure.*

ARTERIES OF THE CORPUS STRIATUM.—*Ch*, Chiasma; *B*, Section of the optic tract; *L*, Lenticular nucleus; *I*, Internal capsule; *C*, Caudate nucleus; *E*, External capsule; *T*, Claustrum; *R*, Island of Reil; *V*, *V*, Section of the lateral ventricle; *P*, *P*, Anterior pillars of the fornix; *O*, Grey substance of the third ventricle.

VASCULAR AREAS.—I, Anterior cerebral artery; II, Middle cerebral artery; III, Posterior cerebral artery.—1, Internal carotid artery; 2, Sylvian artery; 3, Anterior cerebral artery; 4, 4, External arteries of the corpus striatum (lenticulo-striate artery); 5, 5, Internal arteries of the corpus striatum (lenticular arteries). The opto-striate artery is not represented in the figure.

lenticulo-striate artery (*Fig.* 22, 4) is the one most liable to rupture. If the hæmorrhage is small it may lodge between the external capsule and the lenticular nucleus, and give rise to no symptoms. If it remain limited to the lenticular nucleus, the hemiplegia which follows the attack is completely recovered from. But if the internal capsule is ruptured (*Fig.* 23), the

fibres below the seat of lesion undergo descending sclerosis, and the paralysis remains more or less permanent. The patch of sclerosis is found in the middle third of the crusta (*Fig.* 24), the longitudinal bundles of the pons, the anterior pyramid of the

FIG. 23.

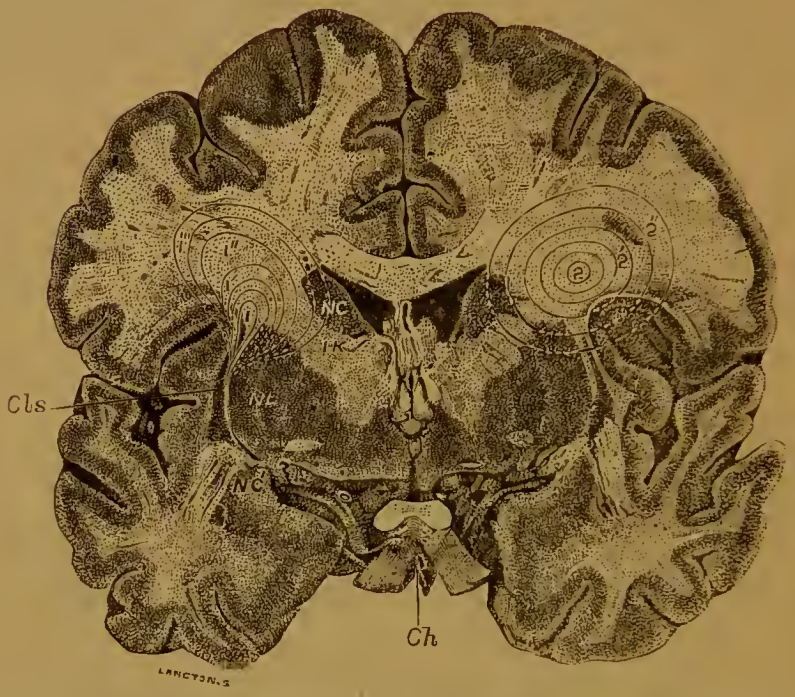


FIG. 23 (modified from Charcot). *Vertical Section of the Brain a little behind the Knee of the Internal Capsule*, showing the effects of rupture of the lenticulo-striate artery.—*NC*, Head, and *NC'*, Tail of the caudate nucleus; *Ch*, Chiasma, *NL*, Lenticular nucleus; *IK*, Internal capsule; *Cls*, Claustrum; 1, The most frequent position in which the lenticulo-striate artery is ruptured; 1′, 1″, 1‴, Progressive extension of the hæmorrhage producing compression and rupture of the fibres of the pyramidal tract (hemiplegia); 2, Primary focus in the internal capsule; 2′, 2″, 2‴, Successive extension of the clot.

medulla oblongata, and the column of Türck on the side of the lesion (*Fig.* 25, B), and in the lateral column of the cord on the side opposite to the hemisphere in which the disease is situated (*Figs.* 26, 27, and 28). If the hæmorrhage ruptures into the lateral ventricle, death takes place in a few hours. Rupture of the lenticulo-striate artery gives rise to a hemiplegia in which the arm is more paralysed than the leg or face. The fibres from the motor centres of the face and tongue pass through the knee

FIG. 24.

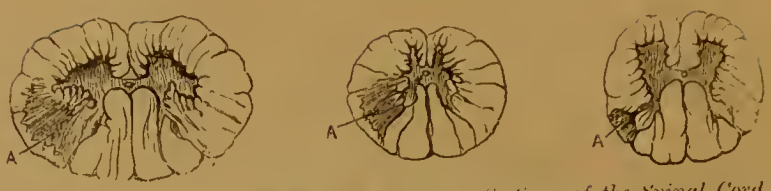
FIG. 24 (Charcot). *Horizontal Section of the Crura Cerebri in a case of Secondary Degeneration.*—*T*, Tegmentum; *F*, Crusta of the healthy side; *L*, Locus niger; *D*, The degenerated fibres, occupying about the middle third of the crusta; *P*, The fibres which undergo secondary degeneration only when the fibres of the anterior segment and the knee of the internal capsule are diseased.

of the internal capsule *(geniculate fasciculus)* (*Fig.* 29, K), and it would seem that those of the arm lie next to them, and those of the leg lie posteriorly near to the sensory fibres, which occupy

FIG. 25.

FIG. 25 (Charcot). *Transverse Section of the Cervical Region of the Spinal Cord, from a case of lesion of the motor area of the cortex of the opposite hemisphere.*—A, Degeneration of the pyramidal tract; B, Degeneration of the direct fibres; C, Direct cerebellar tract; D, Intermediate region between the posterior grey horn and the pyramidal tract, the fibres of which do not undergo descending degeneration.

FIG. 26. FIG. 27. FIG. 28.



FIGS. 26, 27, and 28 (after Charcot). *Transverse Sections of the Spinal Cord, from the middle of the cervical enlargement, middle of the dorsal region, and middle of the lumbar region respectively*, showing descending sclerosis of the pyramidal tract in the lateral column secondary to a cerebral lesion. — A, A, A, Degenerated pyramidal tract.

FIG. 29.

FIG. 29 (after Flechsig). *Horizontal Section of the Brain of a Child nine months of age, the right side being at a somewhat lower level than the left half.*—*F*, Frontal, *TS*, Temporo-sphenoidal, and *O*, Occipital lobes; *Op*, Operculum; *In*, Island of Reil; *Cls*, Claustrum; *f'''*, Third frontal convolution; *Th*, Optic thalamus; *NC*, Caudate nucleus; *NC'*, Tail of caudate nucleus; *LN*, Lenticular nucleus; *I*, *II*, *III*, First, second, and third divisions of the lenticular nucleus; *EK*, External capsule; *IK*, Posterior division; *IK'*, Anterior division, and *K*, Knee of the internal capsule; *ah*, *ph*, Anterior and posterior horns respectively of the lateral ventricles; *gcc*, Knee of the corpus callosum; *sp*, Splenium; *mc*, Middle commissure; *f*, Fornix; *sl*, Septum lucidum; *a*, Cornu Ammonis.

the posterior third of the posterior segment of the internal (*Fig.* 29, IK) capsule. Rupture of the lenticulo-optic artery gives rise to a hemiplegia, in which the leg is more paralysed than the arm, and in which the motor paralysis is accompanied by a hemianæsthesia, but without much implication of the special senses.

Alternate Hemiplegia.—A lesion situated in the crus cerebri will paralyse the third nerve on the side of the lesion, and the

FIG. 30.

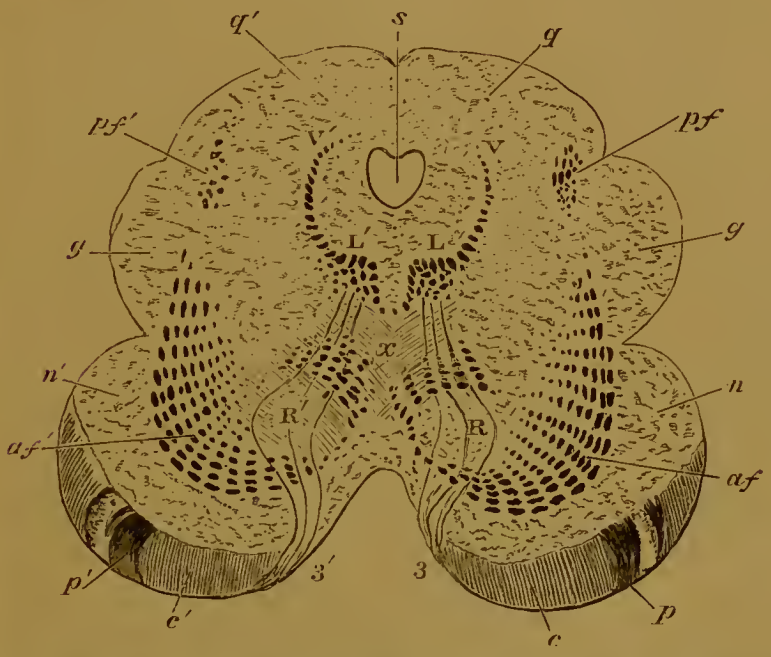


FIG. 30. *Crura Cerebri.*—Transverse Section of the Crura Cerebri on a level with the anterior pair of the Corpora Quadrigemina: from a nine-months human embryo. The dark portions represent Medullated Fibres. *s*, Aqueduct of Sylvius; *q*, *q′*, Anterior pair of Corpora Quadrigemina; *pf*, *pf′*, Fasciculi of Medullated Fibres proceeding to the anterior pair of Corpora Quadrigemina; L, L′, Posterior Longitudinal Fasciculi; V, V′, portions of these Fasciculi which join the posterior commissure of the third ventricle; *g*, *g′*, External Geniculate Bodies; *af*, *af′*, Anterior portion of Fillet; *n*, *n′*, Substantia Nigra; R, R′, Red Nuclei; *p*, *p′*, Pyramidal Tract; *c*, *c′*, Crustæ; 3, 3′, Third pair of Nerves; *x*, Decussation in front of the Aqueduct of Sylvius, which is part of the interlacement of the Tegmentum.

limbs on the opposite side, because the pyramidal tract (*Fig.* 30, p and p′) is injured in its way through the crusta and before its fibres have crossed. If the lesion is situated in the lower part of the lateral half of the pons there is facial paralysis on

the side of lesion, and paralysis of the limbs on the opposite side. If the upper part of the lateral half of the pons be the seat of the lesion, the facial paralysis is on the same side as the paralysis of the extremities. In order to account for this phenomena it is necessary to assume that the fibres of the pyramidal tract, which connect the cortex of the opposite hemisphere with the nucleus of the facial nerve in the upper part of the medulla, cross over about the middle of the pons, as represented in *Fig.* 31.

Fig. 31.

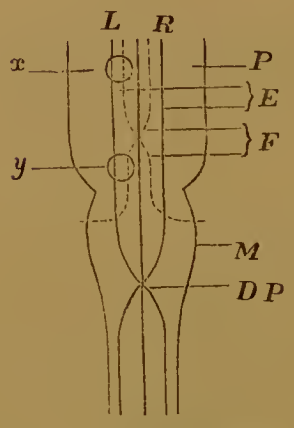


Fig. 31 (from Nothnagel).
L, Left.
R, Right.
P, Pons.
M, Medulla oblongata.
DP, Decussatio pyramidum.
E, Nerve fibres for the extremities.
F, Fibres destined for the facial nerve.
x, Lesion in the upper part of the pons.
y, Lesion in the lower part of the pons.

In *Hemiplegia and Hemianæsthesia, with or without Hemianopsia, Præ-hemiplegic and Post-hemiplegic Chorea, and Unilateral Athetosis,* the lesion has most frequently been found in the area of distribution of the posterior external optic artery. The symptoms do not appear to be caused by lesion of the optic thalamus itself, but by injury of the sensory peduncular and pyramidal tracts, and of the upward continuation of the optic tract as its fibres pass through the thalamus to reach the cortex of the occipital lobe.

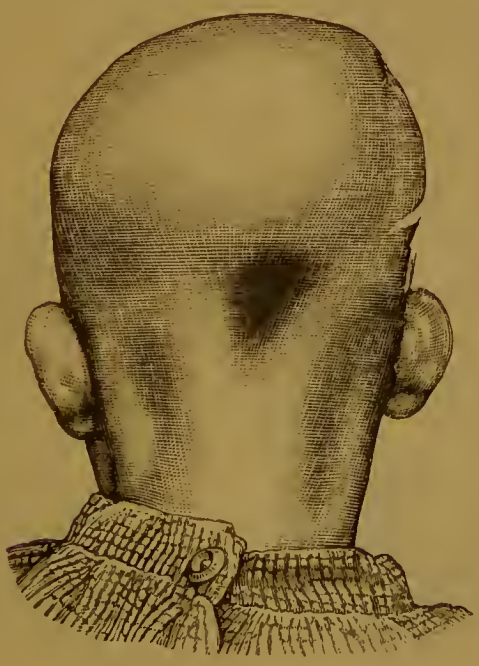
The *Acquired Spastic Hemiplegia of Infancy* is caused by a diffused atrophy of the motor area of one hemisphere of the brain, which is accompanied by a descending sclerosis of the corresponding pyramidal tract.

Congenital Spastic Hemiplegia of Infancy and *Double Athetosis* appear to be the result of a congenital defect of one or both of the hemispheres of the brain, which has been named *Porencephalus.* In one case of this kind which came under my observation there was a defect of the parietal bone on the side opposite to the paralysis, and doubtless there was a defect of the brain at the corresponding point (*Fig.* 32).

When both hemispheres are affected the limbs on both sides of the body are more or less paralysed. In a case of this kind which

came under my own observation, and in which all the limbs were the subjects of partial paralysis and contracture, a cavity filled with serous fluid was found in each hemisphere. It occupied the position of the sulcus of Rolando and communicated with the lateral ventricle (*Fig.* 33). The two central convolutions were wanting on each side. It appeared to have been caused by an arrest of development, and the lateral columns of the cord though small were not in a state of sclerosis. In most cases, however, the cavity is caused by a destructive process such as the occlusion of a blood-vessel, and in such cases sclerosis of the lateral columns of the cord has been observed.

Fig. 32.



In Unilateral Convulsions with Hemiplegia the lesion is situated in or near the motor area of the cortex of the brain. The most common lesions which cause these convulsions are localised meningo-encephalitis, tubercle, syphilitic gummata and other tumours, cicatrices of wounds, and spicula of bone, and of these syphilitic gummata are by far the most frequent lesions. The tissues in the immediate neighbourhood of the main focus are maintained in a state of irritation, and are con-

sequently supplied by an unusually large quantity of blood. The ganglion cells of the cortex absorb an undue supply of nourishment, and discharge themselves in a sudden and explosive manner, thus giving rise to the convulsions. Each cortical discharge is followed by exhaustion, and during this period the muscles which were involved in the convulsions are paralysed.

III. MIXED PARALYSES.

The mixed or indiscriminate forms of paralysis comprise a large class which can only be diagnosticated by those who have an accurate knowledge of the simple forms. It must suffice here to enumerate a few of the many diseases in which paralysis occurs as a prominent symptom, but associated with numerous sensory, vaso-motor, and nutritive disorders. These

FIG. 33.

are acute and chronic myelitis, meningo-myelitis, spinal pachymeningitis and leptomeningitis, hæmorrhage of the spinal cord, spinal meningeal hæmorrhage, concussion, anæmia, and hyperæmia of the spinal cord, cerebral pachymeningitis and leptomeningitis, tubercular spinal and cerebral meningitis, cerebro-spinal meningitis, cerebro-spinal multiple sclerosis, progressive general paralysis of the insane, and numerous other affections.

B. FUNCTIONAL PARALYSES.

Functional diseases are those in which our present means of research do not enable us to detect structural changes corresponding to the functional disorder, and it is therefore manifest that many, if not all of the diseases at present occupying this category, will be transferred to the structural affections as our methods of research become more delicate.

(1) *Toxic Paralyses.*—Many of the forms of paralysis caused by a poison circulating in the blood really belong to one or other of the structural varieties of paralysis already described. There is probably not one form of paralysis hitherto described but may be caused either directly or indirectly by the syphilitic poison, and we have already described diphtheritic paralysis along with the neural paralyses, and lead paralysis along with the spinal atrophic paralyses. Chronic poisoning by mercury causes a paralysis with tremor, which is very similar to the symptoms of sclerosis in patches, while arsenic and phosphorous may give rise to a paralysis of the extremities, which will probably be found to be due to a neuritis of the peripheral nerves. Many drugs, however, cause paralysis in the absence of any recognisable lesion after death. Of these, curara may be taken as a type ; it produces a profound paralysis of all the voluntary muscles by abolishing the conducting power of the terminal nerve endings, but without giving rise to any recognisable lesion.

Alcoholic Paraplegia deserves special mention, inasmuch as it is liable to be mistaken for locomotor ataxia and chronic atrophic spinal paralysis. Alcoholic paraplegia is particularly liable to occur in females; it begins by loss of motor power in the lower extremities which is accompanied by pains in the limbs, and the slightest pressure over the muscles causes great pain, while in some cases there is cutaneous anæsthesia. The patient often experiences difficulty in maintaining her equilibrium, and when she walks the gait is staggering, and the movements of the upper and lower extremities are affected in an irregular and uncertain manner. In a case that came under my observation the patellar-tendon reactions were absent, and the chief features of the disease were very like locomotor ataxia.

(2) *Febrile and Post-Febrile Paralyses.*—The various forms of paralysis which occur in the course of and immediately after the specific fevers and other acute diseases are generally caused by some organic lesion, and they are consequently comprised under one of the varieties of neural, spinal, or cerebral affections already considered. In other cases the paralysis is neither accompanied by atrophy nor spasm, and after a time complete recovery takes place, so that it is most probably caused by general nutritive changes, without any definite localised lesion. During an attack of intermittent fever the patient may be seized with an extensively distributed paralysis which disappears rapidly after the cessation of the attack, or is readily curable with quinine, and such a paralysis must consequently be regarded as of functional origin.

(3) *Reflex Paralysis.*—This form is caused by irritation of the urinary organs, uterus, intestinal canal, or some other of the viscera, and generally appears in the form of an incomplete paraplegia. The cutaneous reflexes are said to be increased; there is complete absence of pain in the loins, girdle pains, dysæsthesia, anæsthesia, muscular tension and contractures, paralysis of the bladder, bed-sores, and other trophic disturbances, the muscles do not undergo atrophy and give normal electrical reactions, and a rapid improvement occurs on the removal of the peripheral source of irritation. In some cases the peripheral source of irritation gives rise to an *ascending neuritis and secondary myelitis*, and in such cases the organic affection of the spinal cord, which is usually a transverse subacute myelitis, must be recognised by the usual tests. Disease of the joints is sometimes followed by paralysis with atrophy of the extensor muscles, which may sometimes persist long after the joint affection is cured. In such cases the mechanical irritability of the affected muscles is in excess, the tendon-reactions are exaggerated, and the electrical reactions are only diminished, and do not usually manifest qualitative changes, while abnormally energetic contractions are obtained by insulating the patient and passing sparks of static electricity through the paralysed muscles. The feebleness and emaciation of the deltoid which occurs in chronic disease of the shoulder-joint, and

the flattening of the buttocks from wasting of the gluteal muscles in hip-joint disease, are good examples of this affection. The paralysis is most probably caused by irritative changes in the spinal cord occasioned by the local disease, but which do not amount to a decided myelitis.

(4) *Post-Epileptic Paralysis.*—After an attack of unilateral epilepsy the muscles which were implicated in the spasm are found to be partially paralysed, and after attacks of general convulsions all the voluntary muscles are found to be affected by a general feebleness which is undoubtedly of the nature of a paralysis. The excessive discharge from the motor centres of the cortex which occasioned the spasm during the convulsive attack is followed by exhaustion and more or less of paralysis results until the activity of the centres are restored by nourishment. Chorea is sometimes accompanied by some degree of paralysis.

(5) *Hysterical Paralysis.*—Hysterical paralysis generally simulates some form of spasmodic paralysis and consequently the tendon-reactions are exaggerated, and even ankle-clonus may be elicited; the muscles do not undergo atrophy and the electrical reactions are normal. In *hysterical paraplegia* the paralysis is seldom complete, the patient being able to move her limbs in bed with comparative ease; she may even be able to get out of bed, but after walking two or three steps the limbs give way, the gait becomes tottering, and the patient falls unless supported by her legs becoming doubled up under her. In walking a hysterical patient glides the passive foot along the floor, and the toes do not scrape the ground as in lateral sclerosis. The hysterical patient can elevate the toe from the ground while standing erect, while all efforts on the part of a patient suffering from lateral sclerosis to elevate the toe only fixes it still more strongly to the ground. In hysterical paraplegia the reflex of the sole is diminished, while in lateral sclerosis it is increased.

Hysterical hemiplegia is usually accompanied by well-marked disorders of sensibility; there is no facial or lingual paralysis; the paralysis is scarcely ever complete; in the large majority of cases the leg is more affected than the arm; it is liable to sudden

variations in intensity under the influence of emotions; the electrical excitability is unchanged; and the muscles do not undergo atrophy. The concomitant symptoms of hysterical paralysis are anæsthesia of variable distribution, tympanites, constipation, dysmenorrhœa, retention of urine, and a peculiarly watchful and stealthy expression of countenance.

A. Ireland & Co., Printers, Manchester.